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СИНДРОМ ПОРТАЛЬНОЙ ГИПЕРТЕНЗИИ У ДЕТЕЙ СО СЕРДЕЧНОЙ НЕДОСТАТОЧНОСТЬЮ

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АННОТАЦИЯ

Портальная гипертензия может быть вызвана целым рядом состояний. Часто проявляется кровотечением из варикозно расширенных вен пищевода. Подход к острому варикозному кровотечению у детей представляет собой пошаговое продвижение от наименее инвазивного к наиболее инвазивному. Лечение острого варикозного кровотечения несложное. Но данные о первичной профилактике и длительном лечении рецидивирующих кровотечений из варикозно расширенных вен у детей недостаточны, поэтому для установления передовой практики необходимы проспективные многоцентровые исследования. Данный синдром часто сопровождается при правожелудочковой сердечной недостаточности, которая вызывается констриктивным перикардитом.

Ключевые слова: портальная гипертензия, варикозное кровотечение, дети, сердечно- сосудистая недостаточность.

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PORTAL HYPERTENSION SYNDROME IN CHILDREN WITH HEART FAILURE

ANNOTATION

Portal hypertension can be caused by a variety of conditions. It often manifests as bleeding from varices in the oesophagus. The approach to acute variceal bleeding in children is a stepwise progression from least invasive to most invasive. The treatment of acute variceal bleeding is straightforward. But data on the primary prevention and long-term treatment of recurrent variceal bleeding in children are scarce, so prospective multicentre studies are needed to establish best practice. This syndrome is often associated with right ventricular heart failure, which is caused by constrictive pericarditis.

Keywords: portal hypertension, variceal bleeding, children, cardiovascular failure

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YURAK YETISHMOVCHILIGI BO'LGAN BOLALARDA PORTAL GIPERTENZIYA SINDROMI

ANNOTATSIYA

Portal gipertenziya bir qator holatlar tufayli yuzaga kelishi mumkin. Ko'pincha qizilo'ngachning varikoz tomirlaridan qon ketish bilan namoyon bo'ladi. Bolalarda o'tkir varikoz qon ketishiga yondashuv eng kam invazivdan eng invazivga bosqichma-bosqich o'tishni anglatadi. O'tkir varikoz qon ketishini davolash oddiy, ammo bolalarda takroriy varikoz tomirlaridan qon ketishining birlamchi oldini olish va uzoq muddatli davolash to'g'risidagi ma'lumotlar yetarli emas, shuning uchun ushbu kasalikni o’rganmoq uchun ko’p izlanishlar zarur. Ushbu sindrom ko'pincha konstriktiv perikardit tufayli kelib chiqadigan o'ng qorincha yurak etishmovchiligi bilan birga keladi.

Kalit so'zlar: Portal gipertenziya, varikoz tomirlari, bolalar, yurak-qon tomir yetishmovchiligi.

Congenital heart defects represent 0.8% of all newborn children [1]. A separate category of study is heart defects with functionally single ventricle (FSVC). Radical, biventricular correction in this category of patients is impossible; optimal results are provided by the use of staged hemodynamic correction [1]. The first stage - with increased pulmonary blood flow - pulmonary artery narrowing, or with decreased pulmonary blood flow - systemic pulmonary anastomosis (modified Blok-Taussig shunt (MBTSh)). The second step is a bidirectional cavopulmonary anastomosis (BCPA). The third step is total cavopulmonary anastomosis (TCCA) surgery in the extracardiac conduit version with fenestration, followed by closure of the latter 6-12 months after TCCA surgery [4]. In patients with Fontaine circulation the central venous

pressure has been shown to be higher than normal, correspondingly compensatory pressure increases in the hepatic veins and the portal system, which ultimately leads to changes in the liver parenchyma. Fibrotic changes in the liver are a serious complication of postoperative TCPS in the long term and become one of the causes of mortality [11]. Previously, such complications were thought to occur not earlier than 5 years after total cavopulmonary anastomosis. However, pathologoanatomic studies of the liver in patients of different age groups and in different time periods after TPMS operation showed that complications in the form of the liver fibrous changes can occur in early postoperative period (up to 1 month), and it cannot be excluded that such liver changes can occur even before TPMS operation [6].

Portal hypertension is an increase in pressure in the portal vein system. Normal pressure in the portal vein system is considered to be 5-7 mmHg. Pressures above 10-20 mmHg lead to dilation of the portal vein. High portal pressure with delayed blood flow leads to splenomegaly and hypersplenism, varicose veins of the oesophagus and stomach, dilation of the anterior abdominal wall veins, haemorrhoids, ascites [2]. The main factor causing portal hypertension development is the increasing resistance to hepatic blood flow, resulting in collateral veins dilation and formation of portocaval shunts, which in its turn aggravate the process, leading to decreasing vascular resistance and chronic portal hypertension. A well-known way to assess the presence of fibrotic changes in the liver is a biopsy. It is the gold standard for staging fibrotic and cirrhotic liver changes. This technique has a number of disadvantages: it is invasive and painful, there is a high risk of complications, and it is difficult to assess over time [5,7].

Other noninvasive techniques are known that allow the presence of fibrotic changes to be assessed in the early stages of the process in addition to biopsy: fibroelastography, a type of ultrasound technique that uses low-amplitude ultrasound signals. The test is easy to use, takes less than 5 minutes, and requires no prior preparation. However, there are differences in liver thickness between girls and boys. It is much higher in males than in females [9]. This is due to the difference in fibrogenesis, as female sex hormones inhibit the development of fibrotic changes in the liver.

The disadvantage of this method is that it is low-informative in detecting early fibrotic changes in the liver, besides being expensive and having difficulties in assessing liver parameters in dynamics. Direct manometry of the portal vein (hepatic vein occlusion pressure) and hepatic veins in the radiology department is not routinely performed in children, as this technique is invasive and has a high radiation load. MRI and MSCT are expensive, difficult to perform routinely, and of inferior diagnostic value compared with liver ultrasound [8,12].

The most common and most informative method for diagnosing hepatic changes is hepatic ultrasound angiography. This technique has long been described and is actively used to detect hepatic and spleen pathology. Ultrasound method enables to trace the dynamics of increasing manifestations of portal hypertension at different stages of pathological process development. The method is informative, but in the presence of marked fibrotic changes in the liver, these changes cannot be detected in the early stages of the process. No adequate prototype has been found in the patent and medical-scientific literature under study [14]. The aim of the invention is to create a non-invasive method of diagnosing early hepatic fibrosis in children with a functionally single ventricle. The set problem is solved by application of quantitative ultrasound densitometry - method of liver parenchyma condition assessment according to the ultrasound wave transit rate and their absorption by liver tissue. The liver parenchyma is measured in 3 different points: non-vascular zone (parenchyma), vascular zone and capsule, for which the convex sensor is placed in the right subcostal area at the level of 7 and 8 liver segments, visualization is performed in V-mode with frequency of 5 MHz and depth of 5-8 cm, the size of the studied area is 5 × 5 mm; when value of density of liver tissue to liver parenchyma exceeds 17 dB, presence of hepatic fibrous changes is diagnosed [15]. Density values in the liver capsule and in the vascular zone are determined to level out the measurement error and to enable further correction of the liver tissue true density in the liver parenchyma zone, proceeding from the density values of the first two zones.

The advantage of the method proposed as an invention is that it is noninvasive, does not require additional preparation, the whole examination takes no more than 5 minutes, and most importantly, it is possible to control the liver parameters in dynamics [18].

The distinctive features have shown in the claimed set of novel properties that are clearly not derived from the state of the art in the field and are not obvious to the specialist.

The method is carried out as follows.

The method is performed on the device (ultrasound system) IE-33 X-Matrix (Philips) using convex transducer C5-1. Apart from standard measurements of ultrasound indicators of the liver (CRC, RDC, blood flow in inferior vena cava, blood flow in portal vein), quantitative assessment of the sound-absorbing structure of the liver (quantitative ultrasound densitometry (QUDM) was used, allowing to avoid presence of such artifacts as acoustic (noise) shadows and excluding blood flow in liver vessels from the area of interest [10].

In quantitative ultrasound densitometry, the registration probe lubricated with contact hypoallergenic gel is placed in the area of the right subcostal region (at the level of 7 and 8 liver segments). The visualisation is performed in the B-mode with the frequency of 5 MHz and depth of 5-8 cm, the size of the investigated area is 5 × 5 mm. A densitometer is used to record the characteristics of ultrasound waves with further data processing. All necessary information about the patient (age, sex, height and weight) is entered into the database. Saving of the data obtained is done in the DICOM program, which allows estimating the density of the liver tissue of each patient in the dynamics [9].

The examination is performed at 3 points: the capsule (the densest structure of the liver): the vascular zone (the least dense structure) and the non-vascular zone (the parenchyma). The characteristics of the ultrasound wave as it passes through the liver change depending on the condition of the liver tissue. Normally, the echo signal from the liver parenchyma is much smaller than the wavelength of a standard ultrasound signal. In the presence of fibrotic changes in the liver, the echo signal becomes visually larger than the standard ultrasound waveform. CUDM considers two main indices: the T-index (the result of comparing the density of the liver tissue of a patient with the reference index) and the Z-index (the result of comparing the density of the liver tissue of a patient with the average index of his age group). More than 50 patients were analysed: normal patients whom we considered as a reference; and patients with altered liver tissue of varying degrees of severity (from minimal to severe (accumulation diseases - glycogenosis)). As the T-index is not used in paediatric practice, we used the Z-index, which was 17 dB in our study [6]. Density values in the vascular zone and liver capsule are measured in order to compensate for measurement errors. As fluids are the least dense media according to DMS, the value in the vascular zone should be 0-1 dB (less than 2 dB). Exceeding the normative value in the vascular zone was considered as a measurement error (overestimation of the true value) and was corrected by reducing the liver parenchyma density by a multiple of the number of dB exceeding the norm in the vascular zone. In contrast, the liver capsule is the densest structure and ranges from 35-45 dB. Exceeding the index of 45 dB was considered as a measurement error and led to the correction of the density index in the liver parenchyma [13].

Thus, the normal liver parenchyma density of a healthy person, irrespective of age and sex, should be between 15-17 dB. In the presence of fibrotic changes, the density of the liver parenchyma begins to exceed the set limit. Children with extrahepatic portal hypertension often also have congenital abnormalities of the cardiovascular system. The combination of cardiac defects and extrahepatic portal hypertension aggravates patients' central and peripheral hemodynamic abnormalities, worsening the course of both diseases and negatively affecting the prognosis. In the diagnosis of cardiovascular pathology associated with extrahepatic portal hypertension, methods of examination of patients with cardiovascular disease are used [2,6]. The optimal treatment for extrahepatic portal hypertension in patients with cardiovascular disease is port-portal bypass surgery, which helps to reduce or terminate the pulmonary arterial hypertension associated with portal hypertension. Children with extrahepatic portal hypertension (EHH) often also have cardiovascular abnormalities. The basis of cardiovascular pathology are

collagen diseases (systemic connective tissue disease), congenital and acquired heart defects (systemic pulmonary shunts, valve defects), disorders of cardiac rhythm conduction. The combination of congenital heart defects (septal defects, i.e., malformations with enrichment of the small circle of circulation) and CHD aggravates the central and peripheral hemodynamic disorders in children, worsening the course of both diseases and negatively affecting the prognosis [2,4]. Purpose of work: the development of diagnostic and therapeutic management of patients with CHD and concomitant cardiovascular disease. It is well known that secondary liver disease can result from congestive heart failure. Elevated inferior vena cava/arterial vein pressure and low cardiac output are considered to be the etiology of secondary liver disease. Severe and prolonged hypoxemia and reduced hepatic blood flow can damage liver tissue. In the aspect of congenital heart disease, this problem is particularly relevant for patients with univentricular haemodynamics. There are studies in the medical literature in which centrilobular (zonal) hepatic necrosis has been identified in coarctation of the aorta, stenosis of the aortic valve, and hypoplasia syndrome of the left heart. Only 2 cases of secondary liver disease (hepatocellular carcinoma) have been described in transposition of the main arteries after Musturd surgery and 1 after correction of tetrada Fallo. The aim of our report is to present an atypical occurrence of extrahepatic portal hypertension in a child 12 years after successful anatomical correction of transposition of the main arteries [8].

Conclusion: liver function should be routinely assessed in all patients with congenital heart disease to improve longevity and quality of life.

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